"Notes on Duodenal Ulcer" -

(Its Symptomatology and Diagnosis)

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INTRODUCTION.

"Duodenal ulcer" proper is also known as "simple duodenal ulcer" and as "peptic duodenal ulcer." A distinction should be drawn between this and that form of ulceration due to tuberculosis, enteric fever, chronic nephritis, septicaemia, cardiac disease, pneumonia, cancer and ulceration following burns involving a large surface of the skin.

The subject has come much to the fore of late, and ulcer of the duodenum is perhaps at the present moment the most discussed abdominal condition.

Apparently about twenty years' ago it was looked on as a very uncommon disease, as Perry and Shaw (Guy's Hospital Rep., 1893)[1] were able to collect all the cases recorded up to that time in which a definite diagnosis of ulceration of the duodenum had been established.

How can its apparent frequency of occurrence now-a-days be explained? Duodenal ulcer, though it cannot be considered a very common disease, is undoubtedly not a rare condition at the present time. Presumably, cases of duodenal ulcer have not increased in number during the last few years; and we may therefore assume that, as a result of more perfect methods of diagnosis, cases are now recognised which were/

were formerly overlooked.

We will now endeavour to reconcile this assumption with actual facts and figures culled from the available literature at our disposal.

Perry and Shaw (Guy's Hosp. Rep., 1893)[1], from an analysis of 17,652 postmortem reports (1826 - 1892), concluded that duodenal ulcer was present in 0.4 per cent of cases. The Fenwicks' researches ("Ulcer of the Stomach and Duodenum", Lond., 1900)[2] proved the existence of an open ulcer of the duodenum in 0.26 per cent of 13,055 necropsies. Chas. F. Martin ("A System of Medicine" by Osler and McCrae, Vol. V, p.207, 1909)[3] says that "pathologically these ulcers are found in 0.297 per cent of all antopsies."

The relative proportion of gastric to duodenal ulcers is variously given as 9 to 1 by Trier ("Ulcus corrosivum duodeni", Copenhagen, 1863)[4], as 10 to 1 by the Fenwicks,[2] and as 40 to 1 by Andral (Allbutt and Rolleston's "System of Medicine" 1907, Vol. 3, p.556)[5]. However, well-directed surgery plus improved diagnosis have combined to lower this difference in proportion, which at the present day may be taken as 1 to 1. There is an apparent increase in the proportion of duodenal to gastric ulcer, which increase W. J. Mayo("Brit. Med. Jour.," 4th Nov.,1906)[6] says is due to the fact that a more definite classification/

classification has been made anatomically. It is now acknowledged that 95 per cent. of all duodenal ulcers reach up to the pylorus or within $\frac{3}{4}$ inch of it, while formerly all ulcers in the vicinity of the pylorus were put down as pyloric and ipso facto gastric. It should also be borne in mind that, as adhesions alter normal appearances, the above points were tardily recognised.

An ulcer of the stomach and one of the duodenum may coexist; the Fenwicks' figure being 1.7 per cent, while Moynihan and Mayo give it as their opinion that one half of the cases of duodenal ulcer have a coexistent gastric ulcer, ("System of Medicine" by Osler & McCrae, Vol. V, 1909, p.389)[3]. In 44 cases reported by Moynihan in 1902 of ulcer of the duodenum he proved the existence of a gastric ulcer as well, while Mayo's proportion was even greater, ("System of Medicine" by Osler & McCrae, Vol. V, 1909, p.209)[3]. Dietrich ("Munch. Med. Woch.," March, 1912)[7] states that in 8,538 autopsies at the Hamburg Hospital, during the years 1908-1911, 1.1 per cent showed evidences of gastric ulcer and of haemorrhagic erosions, and 0.4 per cent of duodenal ulcer. The duodenal ulcer was single in twenty-four cases, and multiple in ten; while gastric ulcer was coexistent in four instances, and haemorrhagic erosions of the stomach in two.

Connected/

Connected with the recent work on duodenal ulcer has arisen a discussion as to the exact significance of "hunger pain" and its relationship to duodenal ulcer. Some, mainly surgeons, as Moynihan[8] and his disciples, contending that the pain, which recurs, termed "hunger pain", is practically pathognomonic of duodenal ulcer. The use of this term "hunger pain" is of more or less quite recent origin, and is said to be characteristic of duodenal ulcer, occurring as it does in about two and a half to four hours after the ingestion of food and lasting for some time; its onset varying with the quality and quantity of the food (Chvostek "Das einfache duodenal Geschwur", 1883)[9]. Probably the first use of the expression "hunger pain" was made by Mayo Robson in the "Medical Annual" of 1906 (p.479)[10]. His description is as follows - "Duodenal ulcer is, I feel sure, a much more common disease than is generally appreciated, for some of the patients affected with this disease appear at times to be unusually hearty, their only complaint being that of a "burning" sensation, three or four hours after meals, that may be termed a "hunger pain", since it is relieved by taking food. The patients are frequently wakened by these sensations about one or two in the morning, and have either to take a dose of carbonate of soda or a drink of milk to relieve their discomfort, yet at any moment/

moment they may be attacked by perforation or haemorrhage."

Others, including Hertz, do not ascribe the same significance to hunger pain, and will not definitely diagnose duodenal ulcer unless haematemesis and melaena be present, which symptoms Moynihan ("Duodenal Ulcer", 1912, p.124)[11] says are to be regarded as complications to be foreseen; being testimony of deep ulceration, which should have been recognised earlier.

The importance of all this lies in the question of treatment, as it is now indisputably recognised that, given a case of undoubted duodenal ulcer, which does not respond to medical treatment within a reasonable time, the best treatment is gastro-enterostomy.

In the present thesis I shall endeavour to bring forward clearly the views of the various authorities who have dealt with ulcer of the duodenum, to sum up the evidence in support of or against these views, and finally to illustrate the chief clinical points as met with in a number of cases.

HISTORY of DUODENAL ULCER.

So far as can be ascertained, the first reference to ulcer of the duodenum appears in the "Medico-Chirurgical Transactions" (Lond., 1817, VIII, 232)[12], the author being Mr. Travers, who describes two cases, both in the male sex; the age of one only being given, viz. 35 years (period at which duodenal ulcer most commonly occurs), who also, be it noted, possessed "a strumous habit" (tuberculosis being not infrequently associated with duodenal ulcer). In this particular case, the points of interest are that the patient up to the time of the fatal attack enjoyed "generally good health", and "was seized (whilst dining) with an excruciating pain in the abdomen, unlike any he had ever felt", in the region of the umbilicus and radiating over the entire body, especially the neck and shoulders. The abdomen became hard and tense; respiration "agitated"; while the pulse at that period was hardly affected. "Flatus rose in quantity from his stomach", but there was "no disposition to vomit." Medicine (administered soon after onset of attack) afforded no relief, the patient became "exceedingly restless" and was "unable to bear the slightest pressure of the hand upon the abdomen." Spoonfuls of gruel were "in part returned, as if deglutition was interrupted by/

by a spasm of the oesophagus!" There was no mitigation of the "intolerable anguish", the pulse now became "quick, small and fluttering"; the patient's strength was rapidly exhausted, and, though "his intellect remained clear and perfect", he sank and died thirteen hours after the onset. The autopsy revealed universal inflammation of the peritoneum: adjacent folds of intestine were matted together by recent adhesions; the pelvis contained much bile-tinted fluid; and there was a circular perforation (with peritoneal margin) of the diameter of a writing pen, about two fingers' breadth below the pylorus, and which was the centre of an irregularly shaped ulcer of the duodenum and involved two-thirds of the pyloric ring. The intestinal canal exhibited no other ulceration.

In the second case, there was a history extending over the previous seven years of occasional attacks of "sudden and very violent abdominal pain, always speedily relieved by a wine-glassful of brandy." On this occasion, though in continual pain, the patient had attended to his business (hairdresser), and even walked to the market, with the result that the pain became "intolerable", but recourse to his customary dose of brandy gave no relief. "Now and then he vomited"; and, getting worse, death closed the scene in/

in 36 hours "from the commencement of acute pain." In this instance also, a perforation following duodenal ulcer was found, (of the size of a crow-quill), at the junction of the duodenum with the stomach.

The subject next appears in a resumé by Dr. John Abercrombie in his "Pathological and Practical Research on Diseases of the Stomach" (2nd. edition, Edin., 1830, pp.103 et seq.)[13] of five recorded cases collected by him, viz., one case in 1824 by Irvine of Philadelphia, of a typical chronic tuberculous ulcer; two records by French physicians; and two in the "Midland Medical and Surgical Reporter" of May and November 1829[14]. Dr. Abercrombie also mentions a case as seen in a specimen in the Museum of the Royal College of Surgeons of Edinburgh of perforating ulcer of the duodenum. He makes the sagacious observation that the "leading peculiarity of disease of the duodenum, so far as we are at present acquainted with it, seems to be that the food is taken with relish, and the first stage of digestion is not impeded; but the pain begins about the time when the food is passing out of the stomach, or from two to four hours after a meal." The obvious importance of this remark appears to have been passed over unnoticed for many years until brought to light again within recent date. In 1845, Wunderlich ("Handbuch der Path. und Therap.," III, 175)[15] record-

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the the first case in which a diagnosis was positively made, and thereafter verified at the autopsy. The history is interesting in that the patient, who was a male and aged 51, began in 1845 to complain of pain between the epigastrium and the right hypochondrium, which pain radiated over the entire abdomen. The attacks supervened three or four hours after taking food, and continued until relieved by the vomiting of undigested food a few hours thereafter.. Six months after the onset, the patient vomited in large amount dark blood of foul odour. For about a year after this the pain remained dormant only to return intermittently, with vomiting, until the above symptoms increased in gravity, with emaciation, and death took place in 1852. Briefly, the postmortem disclosed the following condition:-

Stomach enormously distended, with duodenum and pylorus adherent to the head of the pancreas, and a circular perforation (with callous margins) of the size of a farthing was present at the commencement of the duodenum, leading to a cavity about the size of half a walnut, having the pancreas as base.

In 1861, Klinger (of Wurzburg) dealt entirely with perforating ulcer (Archiv. f. Phys. Heilk.," 1861, II, 5)[16]. This was the first treatise on the subject, and contained notes of 3 cases of the author's/

author's and of ten cases collected by him. Following this came two monographs in 1863; respectively by Falkenbach ("De ulcere duodenali chronico", Berlin, 1861)[17], and by F. Trier(Ulcus corrosivum duodeni", Copenhagen, 1863)[4].

The latter is of importance as containing a report of all cases recorded to date, with details of a series of 26 cases (the majority being inmates of the Frederick Hospital, Copenhagen). The "British and For. Med. - Clin. Review" of January 1864,[18] contained an epitome of Trier's paper.

Krauss published his "Das perforirende Geschwur im Duodenum" (Berlin, Aug. Hirschwald)[19] in 1865, in which he gave (for the most part) detailed records of eighty cases. During the interval 1863-1882 several these for the Paris degree appeared, containing no material addition to our knowledge, with the record of a few more cases of duodenal ulcer.

Next came Chvostek in 1883 ("Allg. Wien. Med. Zeitg.," XXVII, 533)[20] with records of 135 published cases and eight cases personally seen by him.

Bucquoy ("Arch. Gén. de Méd.," i, 398 et seq.)[21] in 1887 published for the first time on record his personal diagnosis of five cases solely from the symptoms; one being verified by subsequent autopsy.

We may therefore conclude that Abercrombie (in 1830) and Bucquoy (in 1887) were the first to assert/

assert that a diagnosis of duodenal ulcer could be made during the life of a patient.

Oppenheimer's thesis "Das ulcus pepticum duodenale" (Wurzburg)[22] made its appearance in 1891, embodying a summary, with useful tables of nearly all recorded cases.

In 1894, Collin's valuable thesis, "Étude sur l'ulcere simple du duodenum" (Paris)[23] appeared in 1894, with notes on five cases seen by himself, a summary of 257 cases recorded to date, and details of all cases published during the previous ten years.

A note of appreciation is merited for the thorough account given by Perry and Shaw, viz. "Diseases of the Duodenum", which appeared in the "Guy's Hospital Reports" of 1893[1]. They teem with material of pathological interest, and embody the results of an analysis of 17,653 postmortems between 1826 and 1892.

Up to this period the attention of the surgeon had not been drawn to the subject; for the symptoms of chronic duodenal ulcer were not recognised with enough certainty for the requisite diagnosis, nor was it realised that cases in which haemorrhage or perforation occurred belonged to the province of the surgeon.

In 1894, Mr. H. P. Dean performed the first successful operation on perforating duodenal ulcer ("Brit. Med. Jour.," 1894, i, 1014)[24]. Another successful/

successful case was recorded by Mr. L. A. Dunn ("Brit. Med. Jour.," 1896, i, 846)[25]. A stimulus was now given to the surgical aspect of the question, and these cases were quickly followed by other successful ones.

Weir's address as president of the American Surgical Association ("Med. News", 1900,i,690)[26] contains a full summary of early cases with a critique in particular on perforating ulcer of the duodenum.

A. Codivilla was the pioneer in treating chronic duodenal ulcer surgically ("Sei casi Gastroenterost sperimentale", Mem. Orig. Firenza, 1893, pp. 406 - 421, and "Contrib. alla chirurg. Gastrica", Bologna, 1898)[27]. His first case, aged forty, was operated upon on 22nd March, 1893, and was proved to be one of stenosis of duodenum two fingers' breadth beyond the pylorus. The patient was well five years after. Codivilla operated on his second case on 5th May, 1898. The third case was operated on by Pagenstecher in 1899 ("Deut. Zeit. f. Klin. Chir.," 1899, lii, 569)[28].

Mr. Moynihan of Leeds operated on his first case in January, 1900 ("Lancet", 1905, i, 340)[29], and the surgical aspects of the disease were thoroughly treated of by him in the "Lancet" (1901, ii, 1656).[30]

Other valuable papers were written by W. J. Mayo ("Brit. Med. Jour.," 1906, ii, 1299, and "Jour. Amer. Med. Assn," 1908, ii, 556),[31] and by Codman ("Boston Med. and Surg. Jour.,' 1909, clxi, 313 and 767).[32]

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In the light of present-day knowledge , it is somewhat difficult to understand why ulcer of the duodenum, with its more or less definite symptoms, was not recognised some years earlier than has been the case. Ulcer of the duodenum is now fairly readily recognised and surgical aid generally sought at once, with the result that the number of successful cases is daily increasing, and to this gratifying state of affairs Messrs. Moynihan and W. J. Mayo have contributed very largely.

SYMPTOMALOLOGY.

Cases are met with presenting the following symptoms:- indigestion with a considerable amount of pain and distress; constipation alternating with diarrhoea; a slight yellow tingeing of the sclerotics accompanied by a trace of bile in the urine; all of which may be put down to a recurring catarrhal duodenitis, stopping short of actual obstruction of the common bile duct . Thanks to our present-day knowledge of the subject one may conclude fairly accurately that this condition is probably due to a recurrent acute exacerbation of inflammation in a healed duodenal ulcer, and enquiry may elicit the fact that the patient has subjected himself to some indiscretion in diet, to over-exertion, or to a chill; conditions now recognised as provocative of renewed inflammation in an already present and, it may be, passive ulcer of the duodenum. Duodenal ulcer is most common in the male sex, and between the ages of twenty-five and forty-five years. For many years, the import of the above symptoms was not fully recognised and various terms were used to meet the occasion and to cloak our ignorance, as gastralgia, dyspepsia, nervous indigestion, neurasthenia, hysteria, neurosis of the stomach, etc. Few conditions present symptoms in such a definite order of sequence as does ulcer of the duodenum/

duodenum. There are exceptions, and instances occur in which this orderliness is wanting in that a single symptom may be so prominent as to overshadow or obliterate the significance of others. As a rule, however, the patient will admit, on careful questioning, that his trouble began when he was comparatively young, and that, with remissions it had lasted up to date. In the early stages there is a feeling of discomfort, the timing of which cannot be definitely fixed. However, it sooner or later becomes apparent that this initial discomfort has advanced to a condition of actual pain and that it is noteworthy in bearing a time-relation to the ingestion of food, by its onset in usually two hours or a little later thereafter. The usual course of events is, complete relief of pain immediately after a meal, only to be followed once more by the onset of the pain and discomfort in two to six hours. A marked feature is the consistency of the interval between the taking of food and the supervention of the pain, provided there is no change in the quantity and quality of the food. Our experience is that when the pain regularly appears in less than two hours after a meal, one of two conditions may account for it - either that stenosis has set in, or that there are recent adhesions with liver or abdominal wall in connection with an active ulcer. On the/

the other hand when the pain comes on rather later, say in three or four hours after food, in all likelihood the ulcer is adherent posteriorly. We have also noted that, when the diet consists entirely of fluids, the onset of pain is proportionately earlier. Generally the pain increases insensibly, accompanied by sensations of being distended and full, and in course of time the pain is described variously as burning , scalding, boring or gnawing. Often there is eructation of gas or regurgitation of acrid fluid, which may afford temporary relief, though at the same time it is commonly complained that the bringing up of this fluid burns or scalds the throat and sets the teeth "on edge". It is usual for a patient to state that the onset of pain is contemporaneous with the feeling of hunger - hence the term "hunger pain" - and that the pain has been always most conspicuous after a heavy meal. On this account sufferers will sometimes desist from taking solid food for months and years. The patient will further confirm our suspicions by saying that he is cognisant of the fact that every meal is followed by pain after an interval, to be relieved by the next meal, then the usual interval and renewal of pain, and so on. He will also say that he is wakened by this hunger pain at night, and/

and almost invariably at the same hour, namely 1 or 2 A.M.; and that he never retires to rest without having some nourishment placed at his bedside of which he can avail himself and so obtain the customary relief. The pain may be confined for a long time to the epigastric region, and then felt as passing through the back and round the right flank. Patients often obtain relief from severe pain by pressure. In a few cases the pain is described as spasmodic like cramp or colic. Spasm of the pylorus does occur, producing an evanescent stenosis from contraction of the pyloric muscle, giving rise while present to a feeling of great distension of the epigastrium. These attacks of spasm are the result of reflex irritation from the duodenal ulcer. Such spasms of the pylorus are the forerunners of dilatation of the stomach. If the ulcer, moreover, be situated close to the pylorus, contractions (supplemented by adhesions) may give rise to permanent obstruction. The appetite is generally unimpaired in spite of pain, but the patient refrains from satisfying the same as he knows that the taking of food beyond certain limits leads to an increase in the amount and duration of the pain. Very often the taking of liquid nourishment, entirely, results in the earlier onset and longer persistence of pain, though perseverance in fluids alone is generally/

generally rewarded in the abatement of symptoms if the condition be in its early stage.

Vomiting is exceptional (being present in less than 20 per cent. of cases), is generally the result of stenosis from the healing of an ulcer or ulcers, and is as a rule a late symptom. It is worthy of note that when vomiting is associated with dilatation, the vomiting often takes place at night, while the material brought up may be greater in amount than the intake and of a frothlike and fermented appearance. It often brings great relief, and is on this account purposely induced by the patients.

The outstanding feature in duodenal ulcer is the periodic and regular occurrence of the symptoms constituting an "attack", with intervals in which there is a complete absence of symptoms. In chronic cases, "attacks" are often precipitated by worry, overwork, indiscretion in diet, or exposure to cold or damp; the last-named being the commonest, and it is a recognised fact that patients suffer more frequently in the colder months of the year. It is also on this score that sufferers remain free from their trouble while resident in warm climates, provided they avoid "chills". The duration of an "attack" - which may begin and end abruptly - may vary from one or two weeks/

weeks to as many months or longer, and all trouble may vanish for the time being by the patient going away for a change, with either complete rest or suitable outdoor exercise, while free of business or professional worries. Between attacks all symptoms may disappear, the appetite is good, and food taken with a relish and without any after discomfort, while there is an increase in weight with a general improvement in health; so much so that the symptoms of an attack are often attributed to "acid dyspepsia" or "hyperchlorhydria."

Haemorrhage, generally a late symptom, is at times an early one (if not the first symptom) of duodenal ulcer, and may as a rule be taken as proof of the ulceration having extended far enough to eat into a large vessel, a condition only possible in most cases after the process has continued for months or years. In fact if the amount of the haemorrhage enables one to state positively that haematemesis or melaena is present, it means as a generality that deep excavation has been produced by an ulcer the presence of which should have been established earlier. As regards the frequency of haemorrhage in this disease, Krauss[19] collected seventy cases in which free haemorrhage had occurred in twenty, while Oppenheimer[22] records thirty-four instances in "over 100" cases. In Perry and Shaw's[1] sixty cases with symptoms out of 151 cases/

cases, twenty-three suffered from haematemesis or melaena. Nine had haematemesis and nine melaena, and five had both haematemesis and melaena. According to Nothnagel's "Encyclopaedia" (p.245)[33] "severe haemorrhage occurs in about one-third of cases." Fenwick[2] avers that haemorrhage is met with in 26 per cent of the acute and in 40 per cent of chronic cases. In view of surgical findings, however, the value of these statistics are now doubtful. Moynihan[34] gives 37.6 as the percentage of cases of haemorrhage in his series.

Melaena usually occurs without haematemesis, but if blood be vomited there is generally evidence of the haemorrhage in the faeces also. When melaena alone is present it is presumptive evidence of the lesion being on the distal side of the pylorus; and it is an established fact that duodenal ulcer is located most frequently in the first portion of the duodenum and on its anterior surface. Copious haemorrhage is generally heralded in by an increase in severity of all the symptoms present, such as giddiness, headache, a congested and tense feeling about the nasal mucous membrane and gums, combined with a disinclination to work, obstinate constipation, with aggravation of the digestive troubles. At the onset of the haemorrhage, the feeling of tension may be relieved, but the patient will probably without further warning become prostrate, with/

with weak pulse, shallow breathing, dim sight, and be bathed in cold perspiration - all symptomatic of an internal haemorrhage, which is confirmed by the passage of a dark tar-like stool, or by the vomiting of blood. In other instances, beyond an almost imperceptible feeling of gradually increasing weakness, the fact that haemorrhage is taking place is not recognised until regular and systematic examinations of the stools prove the presence of traces of blood ("occult blood"), thus establishing with other symptoms the existence of active ulceration. In a small percentage of cases, without premonitory symptoms, death has been dramatically sudden as the result of erosion of a large vessel (in all likelihood rigid and non-contractile). Such cases are termed "latent", in that ulceration may have been present for a considerable period without definite symptoms, until its existence has been revealed by the advent of sudden haemorrhage. The vessels which may be involved are the gastro-duodenal, hepatic, pyloric, superior pancreatico-duodenal, right gastro-epiploic or even the aorta; or the superior mesenteric or portal veins.

An interesting example of haemorrhage occurring suddenly, without any previous warning of the presence of a duodenal ulcer, was related to me by Mr/

Mr. H. J. Stiles. The patient was L't Col. H.B.M. of my Service, on whom Mr. Stiles had operated for haemorrhoids, from which the patient was convalescing, when Mr. Stiles received an urgent telephone message to proceed forthwith to his nursing home. On arrival there he discovered that the patient had, after a slight colicky pain, passed a characteristic "tarry"-coloured motion. There was no other definitely tangible symptom, but with the patient's consent, Mr. Stiles performed a laparotomy without delay, being rewarded by the discovery of a duodenal ulcer, for which a gastro-enterostomy was performed. L't Col. H. B. M. returned to duty in India shortly after, and continues to enjoy excellent health (six years having elapsed since the operation.

On the other hand perforation may be the first tangible symptom, accompanied by shock and rapidly supervening peritonitis. Cases of perforation occurring/

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occurring in patients of previous good health are very rare, though there are records of such having been operated on by Caird, Moynihan, Miles, Mitchell etc. Half of the fatal cases of duodenal ulcer may be put down to perforation, the most serious complication in connection with this condition. Perforation is accompanied by sudden and agonising pain, which may kill the patient forthwith. Should the sufferer withstand the initial shock, his expression is one of deep anxiety, he is pale and bathed in cold perspiration; his respiration is quick and shallow, almost spasmodic; his pulse, at first about 80 per minute, gradually rises in rate but becomes poorer in quality and is an all-important index; the abdominal wall is tense and retracted(before distension sets in),while the patient will flinch even at the weight of the hand placed thereon. Rarely, one may be able to detect a spot of particular tenderness above the umbilical level and slightly to the right of the middle line.

In cases of leakage into surrounding adhesions, a symptom of some value as affording a clue to the same is pyrexia associated with pain during a relapse. Ulcer of the duodenum is, as a rule, unaccompanied by any pyrexia; the tongue too is generally clean, in spite of digestive trouble. That haemorrhage and perforation may occur together has been substantiated by/

by R. J. M. Buchanan, in an address given before the members of the Liverpool Medical Institution in October 1910, with the record of one case seen by him. As to physical signs there may be none very typical of the condition, in spite of the existence, it may be for years, of the symptoms detailed above, and in many cases of duodenal ulcer no information is afforded by an abdominal examination. A tender point may be made out, of two or three inches diameter, in the midline of the epigastric region or slightly to the right, in about two hours or more after a meal, when the pain is worst. At the same time there is usually an increase in the right epigastric reflex, with a firmly contracted condition of the upper portion of the right rectus muscle. Later in the disease, signs of a dilated stomach (with motor incompetence) appear as the result of stenosis. It may, however, occur without any stenosis. Palpation may elicit splashing, and percussion that of an extension of the gastric area, while auscultation (aided by the tuning fork) will define the limits fairly accurately. Dilatation may not always be made evident by radiography following a bismuth meal, though delay in the passage of the food is well shown by use of the X-ray screen. Moreover, in three cases reported by S. Kreuzfuchs ("Wien. Klin. Woch.," No. 11, 1912)[35] traces of bismuth/

bismuth were present in the duodenum for some considerable time, and the patient, when requested to point to any particularly tender spot, pointed to that position behind which the bismuth was visible.

A symptom at times met with in association with duodenal ulcer is the intermittent appearance of tender points under the finger (or even toe) nails, which points become red and finally black. It has not been proved that this sign has any connection with ulcer of the duodenum. As similar points of tenderness occur in cases of infective endocarditis, it is suggestive of the possibility that the condition causing ulceration of the duodenum may be toxic in nature. Cases occur in which severe haemorrhage follows recurrent attacks of these tender points, and simulating Raynaud's disease (short of gangrene).

To sum up; the symptoms of duodenal ulcer may be enumerated thus:- (1) Prolonged or recurrent history of epigastric distress and dyspepsia; (2) Pain, or at least discomfort; (3) symptoms of gastric hyperacidity, (with increased activity of the gastric motor and secretory functions, as demonstrated by the X-rays after a bismuth meal); (4) Blood in the stools.

HUNGER PAIN.

Many theories have been propounded as to the causation of this symptom.

The most generally accepted explanation for many years was that the onset of the pain coincided with the relaxation of the pylorus to permit of the passage of food into the duodenum, pain resulting from the stomach contents causing friction as they passed over the ulcer; and that the administration of an alkali or of food brought about the closing of the pylorus, and, ipso facto, the arrest of the further passage of food over the ulcerated spot in the duodenum and therefore the cessation of the pain.

That this theory was only a conjecture and not founded on facts has been demonstrated by Hertz ("Roy. Soc. Med. Trans.," 1910, and "The Sensibility of the Alimentary Canal", 1911, 59)[36], who in a series of cases administered a meal containing bismuth and then subjected the cases to examination by the X-Rays. He shows that the evacuation of the food from the stomach commences immediately after its ingestion, and that very little of it remains in the stomach when the pain begins two or three hours thereafter. Hertz also notes the increase, as digestion progresses, in the proportion of the gastric juice and of hydrochloric acid in the chyme, from the excessive/

excessive and prolonged secretion of normal gastric juice (the origin of "hyperchlorhydria" in ulcer of the duodenum). In the early stage the major portion of the acid combines with the food proteins and the alkaline salts, and the small amount of free acid passing into the duodenum is quickly neutralised by the alkaline juice of the intestine, the bile, and the pancreatic juice. After two or three hours, however, the presence of a proportionately larger amount of acid results in some of it reaching the ulcer before neutralisation. The contact of acid with the healthy portion of the duodenal mucous membrane brings about arrest of the relaxation of the pylorus, and this inhibition is increased by the presence of the ulcer, resulting in the occasional passage through the pylorus, at long intervals, into the duodenum of small quantities of hyperacid chyme. Neutralisation of the acid by alkalis or proteins, or its dilution by food or water brings instant relief, as also does the removal of the acid and the emptying of the stomach by the act of vomiting or by "washing"; both of which are resorted to by patients, on account of the relief afforded. Our experience also confirms Hertz' statement that there is spontaneous disappearance of the pain when the stomach is entirely empty, that is when there is nothing/

nothing left upon which the muscular coat can contract; and, as the pain apparently supervenes when more than half the meal has left the stomach, I am of opinion that Hertz' explanation of the pain being due to the stomach contents possessing a greater acidity towards the close of the digestive act is sound. The more solid the food and the more peptic digestion required, the longer will the food remain in the stomach. If the food is very irritating, and there is marked hyperacidity, the pylorus will remain closed for some time. It is a matter of experience that pain is most marked when accompanied by a hyperchlorhydria. S. Kreuzfuchs (Wien. Klin. Woch., No. 11, 1912)[35] agrees with Hertz that gastric hypertonus results from ulcer of the duodenum; but, in several cases, he found traces of bismuth still in the stomach six hours after a meal, the duodenum (as a rule) being free from bismuth, and the small intestine to a great extent filled with it. Haudek who had observed a similar condition six hours after a meal, put it down to diminution of the motility of the stomach induced by duodenal ulcer. Kreuzfuchs, as regards the passage of a bismuth meal, differentiates between the fasting condition (when stomach and small intestine are free from food); and the emptied stomach (when/

(when the small intestine contains food); in the former a typical picture of the effect of duodenal ulcer being presented, while in the latter the picture may be quite atypical. The points requiring explanation are:- (1) Increased motor activity of the stomach; (2) Initial rise in the rate of emptying of the stomach; and (3) presence of traces of bismuth six hours later. According to Barclay, the effect of the presence of duodenal ulcer is to largely abolish the duodeno-jejunal reflex , which checks the normal periodicity of opening of the pylorus, and that therefore the pylorus opens more frequently than is normally the case. Experiment supports this view, for by producing a duodenal fistula in a dog and by filling its fasting stomach with an indifferent fluid, the stomach is seen to empty itself at regular intervals (in 18-20 seconds) until all the food is passed on. Hirsch found that this regular action only occurs if the duodenal fistula be not further than 1½ inches from the pylorus, and if the stomach contents empty themselves through the fistula. Apparently injury of the duodenal wall (as in ulcer) acts like the fistula in lowering the duodenal reflex; the real factor is probably therefore not an increase of motor activity but a decrease in the check on the emptying of the stomach. However, according to Kelling, the jejunal reflex/

reflex acts more powerfully as the jejunum fills up, and it may thus be that the duodenal reflex (absent at first) may be awakened by a summation of irritation and exercise a late but energetic action in preventing the rapid disappearance from the stomach of the remaining traces of bismuth.

Kreuzfuchs suggests that hunger pain coincides with the closing of the pylorus. Barclay ("Archives of the Roentgen Ray", 1910, 123)[37] points out that the stomach is sometimes empty in half an hour, and he thinks this rapid emptying may account for the hunger pain. Relief of pain almost immediately after taking food is explicable on the supposition that the setting in motion of the stomach, due to taking food, overcomes the resistance of the pylorus.

The result of X-ray investigation in duodenal ulcer is to emphasise treatment tending to diminish motor activity rather than the treatment of hyperacidity.

Hans Kehr ("Munch. Med. Woch.," June 11 and 18, 1912)[38] states that, according to his observations, hunger pain is the result of a distended gall bladder being bent over by the adhesions connected with a duodenal ulcer. He says - "I have always observed that bile accumulates in the gall bladder only when the stomach and duodenum are empty, viz. when the patient/

patient is hungry. If a gall bladder fistula has been made, bile is secreted almost entirely during the night, when the patient does not eat anything. During the day when regualr meals are given, very little bile flows out, it being at once excreted into the duodenum. I checked this observation experimentally. Colleagues whom I operated upon were so kind as to place themselves at my disposal for experiments. So, for instance, I did a cholecystostomy on a surgeon and in connection with this I noticed that, as in the case of other patients who underwent the same operation, the flow of bile was very small during the day, but exceeding abundant during the night. I asked this colleague to turn the day into night as regards meals. He received his first breakfast at 8 o'clock in the evening, his lunch at 11 p.m., his dinner at 1 - 2 a.m., his after-dinner coffee at 4 a.m., and another meal at 8 a.m., in the morning. He used to sleep during the day till the evening. The flow of bile was reversed: it became abundant during the day and stopped at night. This proves that almost the whole of the bile is consumed, if the patient regularly takes his meals, whereas in case of hunger it accumulates in the gall bladder straining and stretching same. What is the cause of hunger pain? Surely in most cases the fact that the gall/

gall bladder is extremely strained and distended. But if the patient takes some milk or biscuits, the gall bladder is emptied by the reflex from the papilla. At one stroke you thus get rid of the pain. Moynihan and Robson assume that the relief of pain after taking food is due to the free hydrochloric acid getting combined or, in case of liquid food, diluted. Franklin is of the opinion that this is the result of the temporary closing of the pylorus. But we know from what is stated by Krauss and Holzknecht that five minutes after taking food the latter passes into the duodenum. We further know, that the contents of the stomach passing the papilla duodeni causes a flow of bile from the gall bladder, and I therefore think that my explanation is most simple. The hunger pain is a result of the obstructed gall bladder being bent over by the adhesions, which often occur in cases of ulcus duodeni, at least according to my observations."

It is generally conceded that, as a rule, scars in the region of the duodenum do not give rise to pain unless there be adhesions. In the case of an active ulcer, involvement of the peritoneum causes pain, from extension of inflammation along the lymphatics in the neighbourhood of the ulcer.

Though hunger pain is generally a prominent symptom/

symptom, it is by no means pathognomonic of duodenal ulcer, for a very similar and indistinguishable pain is met with in cases of gall stone and inflammation of the appendix. The difficulty can be solved by a laparotomy, after a fair trial of medical treatment (provided there be no urgency), but such an experience lowers our opinion of the diagnostic value of hunger pain. In such cases the physician is confronted with the difficulty of separating organic disease from a purely functional condition, as evidenced by the presence of pain in the later stages of digestion. I am of opinion that very similar pain occurs in cases in which there is hyperacidity, termed "hyperchlorhydria." That such cases do exist has been proved by the demonstration after a test meal of the presence of an excess of acid, and when an operation is (by error) performed, of the non-existence of an ulcer in the duodenum. Ulceration never occurs without leaving some trace, e.g. a scar, of the process, and therefore if hunger pain invariably established the presence of duodenal ulceration, such scars would be oftener found than they are at autopsies, as witness the discovery of only the remains of three duodenal ulcers in 2,000 autopsies at the London Hospital ("Lond. Hospt. Rep.," 1909-1910).[39] From my own experience I have no hesitation in stating that the/

the diagnosis of duodenal ulcer is not such an easy matter as is affirmed by Moynihan, and that hunger pain and hyperchlorhydria are not pathognomonic of duodenal ulcer. These two symptoms may be present, however, as a separate and entirely functional condition. Moreover, surgeons who deal with a speciality, and with material selected for them, are inclined to lose their sense of proportion, and therefore to confuse facts and to consider that the condition they are dealing with occurs more frequently than it actually does.

HYPERCHLORHYDRIA.

The most common of all gastric disturbances. It denotes an excessive secretion of hydrochloric acid, as the result of the introduction of food, and is met with in a large majority of cases of hyperacidity - whether the sequel of direct irritation of the mucous membrane by the food, or of indirect stimulation through the central nervous system (e.g. in mental strain and worry).

An examination of the stomach contents may disclose a hyperacidity, though this is not so frequently the case as the symptoms would lead one to infer; and, in spite of the fact that hyperchlorhydria is a very common symptom in duodenal ulcer, free hydrochloric acid is not always found to be present in excess. There is, however, a great diversity of opinion as to what is meant by excess of hydrochloric acid, some authorities (as Ewald) affirming that any figure above 0.2 per cent. must be considered a hyperacidity, whereas one meets with many cases possessing a greater acidity than 0.2 per cent. without any sign of hyperacidity. But we must remark on the fact that symptoms of hyperacidity do occur in cases of hyperchlorhydria. Generally speaking, however, any increae above 0.2 per cent. means super-acidity, and, as a rule, is accompanied by symptoms of such, though patients/

patients with acidities of even 0.29 per cent present no evidence of gastric disturbance, while others manifest symptoms of hyperacidity who possess no free hydrochloric acid. In fact each person has his or her own degree of gastric acidity, the functional activity of whose stomach is performed with an acidity which may be more than sufficient or quite insufficient in the case of another individual. Consequently, symptoms of hyperchlorhydria will arise when the gastric secretion attains a degree of superacidity by reason of an increase in acid above this normal proportion.

In this connection (hyperacidity), Dr. G. A. Gibson ("Edin. Med. Jour.", April, 1911, p.325)[50] refers to cases under his care in the Edinburgh Royal Infirmary, thus :- "All the cases were undoubtedly cases of duodenal ulcer; the lesions were seen afterwards at operations, and all of them recovered, with the exception of one who died from acute pneumonia. (He found)the total acidity varied from .1 per cent to .26 per cent. The amount of free acid varied between .02 (very small) and .18 (extremely high), so that one man's experience at any rate shows that in undoubted duodenal ulcer the condition of acidity in the stomach varies within wide limits."

My friend, Dr. R. A. Fleming, recently wrote me in the/

the following terms:-

"All my cases had a high HCl factor, and all were relieved by taking food (I am simply referring to non-malignant ulcers)."

The following case related by Moynihan ("Duodenal Ulcer", 1912, pp. 133 & 418)[51] is of a medical man, who had been a sufferer for 20 years, and in whom the "incessant and intolerable acidity" overshadowed all other symptoms. Gastric analysis, after test meals, showed an acidity slightly over half the normal acidity.

"September, 26, 1908. J. L. W. (medical man), aged fifty. Has had stomach trouble for twenty years. Periodic attacks of burning pain coming two to three hours after food, relieved by the vomiting of a small quantity of intensely acid mucus. Always relieved by food or lavage. Twelve years ago a slight attack of haematemesis. In 1901 an attack of acute appendicitis with subsequent appendicectomy. The present attack of pain began in March. He washes the stomach out usually twice in the twenty-four hours; often has to do so in the early morning (2 A.M.).

Operation: A scarred ulcer with central depression the size of a sixpence on the anterior surface of the first part of the duodenum. Stomach and gall-bladder/

bladder normal. Posterior gastro-enterostomy. Infolding of ulcer. Recovery."

Where gastric analysis proves the presence of an excess of free hydrochloric acid, there is generally some pyloric stenosis, whether organic or spasmodic, resulting in dilatation and atony with delay in passage of the stomach contents, giving rise to irritation of the gastric mucosa, with hypersecretion of gastric juice and of acid - in fact a hyperacidity.

Though hyperchlorhydria is practically always present in ulcer of the duodenum, it also occurs in other gastro-intestinal disorders, causing gastric stasis. Delay in the passage of food means fermentation, with production of lactic acid; and we find a condition of hyperacidity in atonic dilatation of the stomach, gastroptosis, constipation, and in kinkings

of/

P. T. O.

of the pylorus and duodenum. Stagnation of the gastric contents also occurs in active ulceration of the duodenum, with perhaps prominent hyperchlorhydria, by reason of a so-called "protective spasm" of the pylorus, which has the effect of delaying the passage of the over-acid chyme into the duodenum.

Hyperchlorhydria, moreover, may only be symptomatic of a secretory neurosis of the stomach, for which there may be no pathological foundation.

The question now arises - "What is the special significance of hyperchlorhydria in ulcer of the duodenum?" We have our indicator, in the clinical history of duodenal ulcer, that chronic gastritis (with remissions and exacerbations, and hypersecretion of acid) is of considerable importance in its development. The difficulty from the physician's point of view is to be in a position to say definitely when this condition of chronic gastritis has culminated in actual ulcer formation. The onset or existence of melaena generally clears the diagnosis; but, unfortunately, ulceration of the duodenum may be present considerably before the appearance of melaena. When therefore is the physician justified in calling in the surgeon to operate? When the gastric symptoms, and the hyperchlorhydria in particular, fail to show improvement after a patient and/

and careful trial of medical treatment.

With regard to gastric analysis; as a preliminary, an Ewald "test meal" is administered, say at 8 a.m., consisting of two ounces of white bread and half a pint of weak tea. The stomach is emptied an hour after by the aid of a soft india-rubber tube, passed into the organ, and the siphonage started by coughing. To ensure results capable of comparison, test meals must be given on an empty stomach, the composition of the meal being constant, while the stomach must be emptied after a given and regular interval. About 30 c.c. of semi-digested material is generally available after the lapse of an hour from the administration of an Ewald meal. The material obtained by siphonage, must then be carefully filtered; when the rate of filtration will be tardy if there be much mucus , and more complete and rapid if much hydrochloric acid be present and the material more or less diffluent. The filtrate is yellow and limpid, with an odour like raw beef; or,if fermentation has supervened and the smell is sour, it is generally found to be due to the presence of lactic acid; butyric acid is responsible for a rancid, while an excess of hydrochloric and acetic acids gives a pungent odour. Bile will impart a green tinge, and blood a red or brown colouration to the filtrate. A small/

small residue left on the filter is proof of good digestion, while a large residue with little alteration in appearance means poor digestion and deficient gastric secretion.

Litmus paper invariably yields an acid reaction, due to HCl (free or combined), to organic acids, or to acid salts. Evidence of the presence of a free acid is established by means of congo-red paper, which turns a blue colour with an uncombined organic or mineral acid.

To obtain the total acidity of the filtrate, 10 c.c. is tritrated until neutralised by the decinormal solution of caustic soda (4 grammes per litre). The result may be expressed in terms of HCl, or by the number of cubic centimetres of the soda solution required to neutralise 100 c.c. of filtrate. If 7.0 c.c. of the soda solution neutralised the 10 c.c. of the filtrate, the total acidity will be 70. Normally, it varies between 50 and 65. A large acidity means an abnormal quantity of hydrochloric acid, and vice versa.

To demonstrate the presence of free HCl, equal quantities (a few drops) of the filtrate and of a solution consisting of phloroglucin (2 parts), vanillin (1 part), with absolute alcohol (30 parts) are mixed together and heated; when, if free hydrochloric/

hydrochloric acid be present, a crimson ring manifests itself; the degree of acidity being in proportion to the density of colour. The test is very delicate, even to detecting as small a quantity as 0.002 per cent. of free HCl.

To determine quantitatively the amount of free hydrochloric acid present, place 10 c.c. of the filtered contents of the stomach in a porcelain dish, and from a graduated burette drop the decinormal soda solution, frequently stirring with a glass rod. Every now and then a few drops should be removed to another dish and tested with the phloroglucin-vanillin solution, and, when this ceases to give the characteristic reaction all the free HCl has been neutralised. Its percentage is determined by multiplying the number of cubic centimetres of soda solution used by the number .0365. It is advisable to perform the operation twice, and take the mean of the two results.

A patient with a history suggestive of duodenal ulcer should have the duodenal contents examined. This may be accomplished by the administration of a few ounces of olive oil after the stomach has been carefully washed out and rendered completely empty. The oil is withdrawn from the stomach in half an hour. It appears to bring about a relaxation of the pylorus, thus/

thus allowing a regurgitation of the contents of the duodenum into the stomach. The mixture of fluid and oil is allowed to stand, after removal, until separation takes place. Examination of this supernatant fluid may yield us several indications as to the condition of the duodenum.

To estimate the condition of the pancreas, the reaction of the urine to the Cammidge test should be noted. Though this test is by no means infallible, the absence of a positive reaction allows us to assume that the pancreas is intact and that any disturbance or obstruction at or near the entrance of the pancreatic duct is improbable. The urine should also be tested for the presence of bile or indican.

The stools should be examined for the presence of blood and of undigested fibres, of crystals, of bile, or of an abnormal amount of fat; while the reaction to litmus should be noted .

"OCCULT BLOOD".

This symptom, which is a very common one in duodenal ulcer, denotes the presence of haemorrhage in such small quantities that the blood cannot be distinguished by the naked eye, but that it is capable of recognition by other tests.

In all likelihood, the ulcer bleeds as the result of friction of its surface. Moreover, the bleeding is as a rule intermittent, and hence the imperative necessity of a regular and systematic examination of the faeces, which is almost invariably rewarded by the discovery of traces of so-called "occult blood." A negative decision as to its presence should never be arrived at until at least three examinations have been made, for proof of its established presence is of great value in diagnosis, prognosis and treatment. It is worthy of note in this connection that intermittent bleedings are suggestive of ulcer while constant haemorrhages generally denote carcinoma. The diagnosis of the latter is doubtful in the face of persistent negative results; and, in cases of ulceration, it is a matter of clinical experience that occult bleeding is most constant before treatment is commenced, and when there is marked pain.

One must exclude all other sources of haemorrhage, such as occurs from the nose, gums, the oesophageal and/

and gastric mucous membranes (in these the bright red blood is easily recognised by the naked eye); and in purpura, haemophilia, scurvy, arterio-sclerosis, enteric fever, and in tabes with gastric crises. Positive results are also obtained in haemorrhagic pancreatitis, malignant tumours (with ulceration) of the gastrointestinal tract, and of the liver or pancreas; such often giving rise to much confusion. Similarly with alcoholic gastritis, polypi, intestinal parasites, simple catarrhal or syphilitic ulceration of the intestine, tuberculosis, haemorrhoids, and anal fissure and fistula. Enquiry should also be made as to whether raw or imperfectly cooked meat has been partaken of just prior to the examination for occult blood, to avoid error.

The examination should be made especially after pain has been complained of. With this view the benzidin test is the most delicate, and is carried out thus - some faecal matter about the size of a hazel nut is mixed with water in a test tube to form a thick emulsion, to which one-third of its volume of acetic acid is added, and the whole shaken. 5 c.c. of ether are added, and the two fluids are gently mixed and allowed to stand, when the ethereal extract separates. Of this extract 2 c.c. are mixed with 2 c.c. of a saturated solution of/

of benzidin in rectified spirit, and 2 c.c. of a solution of hydrogen peroxide. The solution will at once become a deep blue colour if much blood be present; while the colour will be less intense and green if there be a smaller quantity of blood. There are other tests - the Guaiacum; the Aloin ; Teichmann's micro-chemical; and Meyer's Phenolphthalein test - as well as microscopic and spectroscopic tests.

The Guaiacum Test - Take and soften (if need be) one gramme of stool with as few drops of water as possible. Add 4 or 5 c.c. of glacial acetic acid, and mix intimately by rubbing up in a glass mortar. To this add 30 c.c. of ether and shake well, and allow to settle, when the ethereal extract will separate. Decant 1 or 2 c.c. of the clear extract, add an equal quantity of distilled water and shake thoroughly. To this mixture add a few granules of powdered guaiacum resin (a penknife pointful); and when dissolved, the tube is well agitated. Thirty drops of old chemically pure turpentine are added, and the tube and contents placed with a white surface as back-ground. A distinct light blue colour rapidly develops in the upper half of the mixture, remains for a short time, and then gradually disappears/

disappears when blood is present to the amount of one milligramme to one gramme of faeces . The benzidin test is now useful as a control test to the guaiacum test.

The Aloin Test - In this, a few grains of powdered aloin are placed in a clean test tube and 10 c.c. of a 75 per cent alcohol added, forming a yellow solution. The test is carried out with this solution as in the guaiacum method. If blood be present the result is a pink or darker red colour, which may be delayed for 10 or 15 minutes.

Teichmann's Microchemical Test. In this, faeces and sodium chloride crystals are mixed on a glass slide and a cover glass applied; while one drop of glacial acetic acid is placed so as to run under the cover glass. The slide is next heated over a lamp to steam off the acetic acid; and more acetic acid is applied until the fluid assumes a distinct brown colour. (Care is required not to reach the boiling point). Evaporation in the air follows, and examination is made for the crystals of haematin hydrochlorate (haemin), which occur as small red-brown rhombic plates.

Meyer's Test - In which two grammes of phenolphthalein, twenty grammes of potassium hydrate, and ten grammes of zinc dust are boiled together in 100 c.c. of/

of distilled water until complete decolourisation has taken place, when the mixture is filtered and constitutes the reagent. Equal parts of the reagent and of hydrogen peroxide are mixed in a test tube, and to this mixture the faecal solution under test is added (after having been boiled and allowed to cool). The presence of blood is indicated when a carmine to pink colour appears. It is an accurate test, and is sensitive 1 to 1,000,000.

The microscope may be utilised for the rapid examination of material suspected of containing blood, for the presence of blood cells and pigment. Should the result be unsatisfactory, we may use the spectroscope. In this case, a portion of faeces is dissolved with a little water; glacial acetic acid is added; the mixture filtered, and the filtrate shaken up with ether. The presence of blood is proved by the ether becoming reddish-brown, while the spectrum shows the broad absorption bands of acid haematin in the red.

Wherein lies the value of the search for occult bleeding? With regard to prognosis, prominent or increasing positive reactions to the tests applied are symptomatic of an unhealed ulcer, while continuance of bleeding in spite of medical treatment signifies need of surgical attendance. The value of the search for blood from a therapeutic point of view is obvious, while the occult bleeding lessens under appropriate treatment.

DIFFERENTIAL DIAGNOSIS.

Wrong diagnoses are of every-day occurrence; not only in the early, but in the later stages of the disease, especially in the differentiation of duodenal ulcer from cholelithiasis, gastric ulcer, and appendicitis. Nineteen out of forty-nine cases of Moynihan's were found to be duodenal ulcer with perforation and not appendicitis as supposed, ("Lancet", 1901, ii, 1656)[40]. Moynihan ("Duodenal Ulcer", 1912, p. 139)[41] states that in a consecutive series of 100 operations, wherein he made a diagnosis of duodenal ulcer, an error was made in three cases, of which two proved to be gall-stones, and one gall-stones and appendicitis. Earlier in his practice Moynihan acknowledges the much greater difficulty he then experienced in diagnosis. Rowen ("Jour. Amer. Med. Assn", 4 May, 1912)[42] says he has been struck with the uncertainty of finding this condition and of the reasonable certainty of discovering some other pathological change. In illustration, he cites eight cases, which with the exception of one (which came to autopsy), he had seen and treated from their onset. Their summary shows chronic duodenal ulcer without other lesions three times; once ulcer accompanied by chronic appendicitis, and once its symptoms were confused by/

by a marked osteo-arthritis of the spine; another ulcer was found at autopsy. On two occasions Rowen came on unsuspected advanced carcinoma of the stomach and once found chronic adhesive appendicitis.*

Having briefly referred to the difficulty of diagnosis, we may now proceed to enumerate the symptoms etc., by which we may discriminate ulcer of the duodenum from other conditions.

Cholelithiasis. This exhibits the closest mimicry of ulcer of the duodenum, in which latter, however, there is an orderliness in the sequence of events, with its well-defined "attacks" from recognisable causes, its appearance in particular during the colder months, its alleviation by diet, and its immediate relief by lavage or alkalis, resulting in cessation for the time being of all the symptoms. In cholelithiasis we do not find a similarly precise periodicity. Haematemesis and melaena are not commonly associated with cholelithiasis. Moreover, the pain in duodenal ulcer, though perhaps severe is generally bearable; whereas in cholelithiasis it is frequently beyond endurance; also in hepatic colic the onset of the pain is sudden, (as a rule within an hour after food), disappearing as rapidly, and is often accompanied by nausea and vomiting, (bringing no relief though the stomach be thus emptied). In gall-stones/

*See appendix (6), p. 86.

gall-stones there is a tenderness in the right hypochondrium, often coupled with a sensation of pain in the region of the right shoulder-blade (suggestive of impaction in the cystic duct).

Not infrequently in gall-stones, the sclerotics are jaundiced, while a trace of bile may be found in the urine - rarely in ulcer of the duodenum. In gall-stones, also, ague-like fits of shivering and fever occur; which symptom is non-existent in duodenal ulcer.

However, cases present themselves in which it is extremely difficult to arrive at a definite opinion between duodenal ulcer and cholelithiasis. In gall stone some aid is at times obtained by bimanual examination, viz., by pressing the liver and gall bladder forward from the back with the left hand, the right hand being applied to the front of the abdomen, when a sensitive resistance to pressure is felt, especially at the height of deep inspiration. Pressure from the front alone may elicit no response of pain. In duodenal ulcer there is present, during the attacks of pain, a sense of tenderness on pressure on the right side somewhat above the navel, which tenderness can be detected at once even without a bimanual examination and which remains almost the same during inspiration and expiration.

A/

A typical case of this description was that of Mrs. H. C., aged 30, whom I was called in to see in February, 1903, at Bolarum, Deccan, India. I found her rolling about in great pain, with tenderness on pressure in the right hypochondriac region in particular, but slightly diffused downwards. She was bathed in a cold clammy perspiration. As I had previously treated her for dyspeptic trouble off and on, coupled with the fact that on this occasion she obtained some relief of the pain on vomiting, and that a search thereafter for signs of gall stones, bile in the urine and jaundice was unsuccessful, I was greatly puzzled, as also was Colonel C.M.Thompson (whom I called in consultation). As she was a "liverish" subject, appropriate dietetic and medicinal remedies relieved her for the time being. A month or two later she accompanied her husband to the Central Provinces in India; but as similar attacks occurred, with recurring frequency, she came to Europe for treatment. I met her in 1907 in Princes Street, Edinburgh, when I failed at first to recognise her, she looked so well and plump. She informed me that she had consulted an eminent specialist in London, but had derived no benefit. She came to Edinburgh, and by the advice of Dr. Murdoch Brown, had a laparotomy performed by Mr. Alexis Thomson, who removed/

removed several gall stones! She has since enjoyed excellent health.

Gastric ulcer. The differentiation is somewhat easier in this instance, for, among other signs, if the pain is delayed for over two hours after food we may conclude that in all likelihood the ulcer is duodenal. On the other hand, the onset of pain within an hour after a meal betokens gastric ulcer as a rule (in the lesser curvature probably); while if the pain appears between one and two hours, we may assume that the ulcer is probably in the antrum of the pylorus. Moreover, gastric ulcer does not have the seasonal recurrences so characteristic of duodenal ulcer in the damp and cold weather of the year. Other differences are that the ingestion of food usually relieves pain in ulcer of the duodenum, while it gives rise to pain in gastric ulcer. Also, the seat of the pain, though referred to in the midline in both, is sometimes to the left of it in gastric ulcer, but in duodenal ulcer commonly to the right with tenderness on deep pressure. As regards haemorrhage; haematemesis generally occurs alone in gastric ulcer, while both haematemesis and melaena may be present in duodenal ulcer with melaena in larger amount as a rule. Vomiting is seldom met with in duodenal ulcer, but it is common in gastric ulcer. An interesting clinical/

clinical point is that an ulcer of the duodenum gives a more variable history of improvement than does ulcer of the stomach, probably because of the greater tendency in the former to heal where the surrounding food and liquid are more or less constantly alkaline, while in ulcer of the stomach the food and liquid are more or less constantly acid. Whatever the reason, the intervals of improvement of ulcer of the duodenum are very characteristic.

The increase or decrease of the gastric secretions is of no significance here, as hyperchlorhydria occurs in both ulcer of the stomach and of the duodenum, and Moynihan's contention that recurring hyperchlorhydria is a medical term for a surgical condition is without foundation.

Finally, one must bear in mind that duodenal ulcer occurs most commonly in men, while ulcer of the stomach is more often met with in women and young girls. In fact, cases of anaemia, with digestive trouble, particularly in males, should be carefully investigated and kept under observation. *

Appendicitis. In acute form, with its classical symptoms of (primarily) diffuse abdominal pain, in the right iliac region; with rigidity of the abdominal walls, particularly in the region over the appendix/

*See appendix (1), p.80.

appendix; a feeling of nausea with, as a rule, vomiting; rigors (concomitant with or followed by fever); constipation; quickening of pulse rate; plus a consciousness of being seriously ill, are all characteristic enough to prevent a mistake. It is in cases of "chronic appendicitis", the result of a previous acute attack, that the resemblance to duodenal ulcer often results in an erroneous diagnosis. One is aided, however, towards a correct conclusion by the history of a prior attack of acute or subacute appendicitis; with probably some local tenderness and muscular rigidity in the right iliac region, and evidence of adhesions.*

Pancreatitis. Difficulty chiefly arises in differentiating acute haemorrhagic pancreatitis from perforating duodenal ulcer, and careful enquiry into the history of the case is all-important. The sudden and violent epigastric pain, with vomiting and marked collapse, are typical of the onset. The subacute form is recognised by the demonstration of a tumour mass in the epigastrium between stomach and colon suggesting the existence of an exudate in the bursa omentalis. In the acute and subacute forms of pancreatitis, we sometimes find glycosuria and steatorrhoea (evidences of imperfect pancreatic functioning/

*See appendix (2), p.81.

functioning). Diagnosis is then aided by a supplementary and careful examination of urine and faeces. In the absence of a history pointing to gastric or duodenal ulcer or other condition which might produce perforation of the stomach, duodenum, or transverse colon, the sudden onset with intense pain and rapid collapse suggests acute haemorrhagic pancreatitis.*

Malignant Disease of the Pancreas. The chief symptoms of this condition are a gradually increasing jaundice of marked intensity, associated with dilatation of the gall bladder (the distension remaining) - neither of which signs are common to duodenal ulcer (in which the permanently distended gall bladder is never met with). Other important clues to a diagnosis of malignant disease are rapid emaciation, with the presence of a tumour in the epigastric region, and advanced age. Pain, when it occurs, is unlike that occurring in duodenal ulcer in that it has no connection with the taking of food, is continuous and severe in character and is increased by coughing. Later in the disease one is aided by the presence of fat in the stools, and of leucin and tyrosin in the urine.

Carcinoma of the Duodenum presents great difficulty in arriving at a correct diagnosis in its early stages. Chronic ulceration of the duodenum is often the forerunner/

* See appendix (3), p.83.

forerunner of carcinoma (41 to 13), and is similarly more common in men than in women, but the sufferers are generally older in carcinoma. Rolleston gives the average age as 51.6 years in 53 cases (Rolleston in Allbutt's "System of Medicine", Vol. III, p. 578-579)[43] We may class carcinoma of the duodenum, according to its position, thus:-

(1) Situated in 1st portion of the duodenum; suprapapillary.

(ii) " " 2nd " " " circumpapillary.

(iii) " " 3rd " " " infrapapillary.

(i) In suprapapillary carcinoma, the symptoms are very similar to those of malignant disease of the pylorus, such as dilatation of the stomach with vomiting of its pent-up contents, which may contain blood. However, as the gastric mucosa is not involved, free HCl is generally present, while lactic acid and other fermentation products are absent. A palpable tumour can usually be defined in about 50 per cent of the cases, being less mobile and situated more to the right than is pyloric carcinoma.

(ii) Symptoms of carcinoma in the circumpapillary portion vary according to whether the papilla is involved or not. If the papilla be not involved, the/

the symptoms belong to the same category as carcinoma above or below the papilla respectively. With the involvement of the papilla, we have signs of obstructive jaundice, viz., clay-coloured stools, jaundiced skin and sclerotics, large amount of bile in the urine, a congested and perhaps tender liver, with gastric and other troubles due to obstruction to the passage of bile and pancreatic juice. Ulceration may periodically relieve obstruction, thus producing an intermittency in the above symptoms. Suppurative cholangitis may be superadded, with increase of the liver manifestations, rigors, and a blood picture of septic absorption with marked leucocytosis, anaemia, etc. Circumpapillary carcinoma is extremely difficult and often impossible to distinguish from primary carcinoma of the ampulla of Vater (cavity into which the common bile duct and pancreatic duct open), of the lower end of the common bile duct, or of the head of the pancreas.

The tumour may be palpable as an immobile and hard mass, deeply situated.

(iii) Symptoms of gastric obstruction plus vomiting of bile and pancreatic juice are characteristic of infrapapillary carcinoma. The acidity of the gastric juice is neutralised to a certain extent/

extent, but the combined acidity may not be greatly decreased, though no free acid be demonstrable. The firm immobile tumour may be palpable. In all three forms there is marked emaciation and cachexia.*

Ulcer of the Jejunum follows gastro-jejunostomy, is happily of rare occurence, and is supposed to be the result of the action of hyperacid gastric juice entering the intestine. Its site is close to where stomach and intestine have been united. Its more frequent sequence after anterior than posterior gastro-jejunostomy is probably due to the fact that the stomach is not so well drained by the former operation nor the hyperchlorhydria so adequately relieved. Jejunal ulcer is commoner in men than in women. The peritomitis following perforation is usually circumscribed; and Mayo Robson (Osler and McCrae's "System of Medicine", Vol. V., p. 394)[44] is of opinion that generally peritonitis is of more frequent occurrence (in perforating jejunal ulcer) after the posterior operation.

Suppurating Retroperitoneal Glands arising from various causes may simulate duodenal ulcer, particularly when there is epigastric pain and uneasiness, and tenderness when deep pressure is made, but the separation of the two conditions becomes easier when/

* See appendix (4), p.84.

when the constitutional signs (loss of flesh, fever with rigors, leucocytosis, and a rapid pulse) predominate over the local, while the demonstration of a palpable tumour will further aid us in our differentiation.

Cirrhosis of the Liver, in its early stages, is at times difficult to diagnose; but a history of alcoholism with dyspepsia, a tender and congested liver with recurring attacks of pain, some low fever and it may be jaundice, with bile in the urine, and an enlarged spleen betoken this hepatic condition. The diagnosis is not so facile when haematemesis and other bleedings from the stomach and intestines are superadded to the above symptoms, calling for a differentiation between liver cirrhosis and gastric or duodenal ulcer. Although hyperchlorhydria at times occurs, it is not prominent in cirrhosis; while there is no localised tenderness in the epigastrium. Moreover, in ulcer of the duodenum, jaundice seldom occurs and is slight. Finally, the advent of ascites in cirrhosis will clear any doubt.

In addition to the conditions enumerated above, there are others which are occasionally mistaken for ulcer of the duodenum. Prominent among these are the gastric crises in Locomotor Ataxia, which when severe have/

have prompted the performance of a gastro-enterostomy. Such an error would, however, not occur, if care were taken to elicit the following points, viz., the history of syphilis (in 90 per cent. of cases), the lightning pains, girdle sensation, absence of knee jerk, presence of the Argyll-Robertson pupil, bladder trouble, and ataxic gait.

Chronic Dyspepsia. Having set aside all cases originating in inflammatory or organic states of the stomach and duodenum, and those described as neuroses, we turn to the most frequently met with type of gastric dyspepsia, termed "atonic dyspepsia", really a form of slight motor insufficiency (a gastrectasis) resulting from defective muscular tone. In order to differentiate between this and other forms of dyspepsia and that associated with duodenal ulcer, recourse must be had to a careful examination of the stomach by the usual routine methods, by the X-rays after a bismuth meal, and in particular by an examination of the gastric contents after a test meal.

Gastroptosis. Its presence or otherwise is established by the position of the lesser curvature; for, in this condition, the lesser curvature is abnormally low, while the position of the stomach is altered. In simple dilatation the lesser curvature is in its normal/

normal position.

Also, in simple gastroptosis, motor insufficiency is not manifest after a test meal. A further aid in diagnosing this condition is to give the patient 2 ounces of bismuth subnitrate suspended in a pint of mucilage of acacia, and to forthwith take an X-ray photograph, the patient standing and holding his breath (not forced inspiration). An exposure of 15 seconds should suffice; the plate being in contact with the anterior wall and the rays thrown posteriorly. The emulsion of bismuth should be syphoned off immediately after. The position of the two curvatures of the stomach can then be clearly delineated. In all cases associated with indigestion or dyspepsia the above features will all help in discriminating such conditions from duodenal ulcer.

Symptoms of duodenal ulcer are often closely simulated by <u>kinks of the pylorus and duodenum</u> (due to adhesions), and the report of a case by Sir Lauder Brunton ("Roy. Soc. Med. Trans"., 1909)[45], which was by error operated on as one of duodenal ulcer and which turned out to be that of a kink in the duodenum, demonstrates the extreme difficulty in framing a correct diagnosis in such circumstances.

<u>Nervous Dyspepsia</u>. To establish the diagnosis of this/

this common ailment we must primarily exclude all organic conditions. In addition to the absence of all features of organic lesions, we notice that the gastric motor and secretory functions are normal, and that there are symptoms of general neurasthenia coupled with digestive disturbance. Moreover, that the condition is influenced by change of scene etc., and not by the quality or quantity of the food. In cases of doubt, a good plan is to place the patient on an easily assimilated diet such as is given in the third week of treatment of gastric ulcer, and the subjective symptoms carefully watched for about four days. We may now supplement the diet by the addition of vegetables, sauces, fruit and dessert and a comparison made with the symptoms during the first period. Should the symptoms be not aggravated, or on the contrary lessen in severity, the condition we are dealing with is in all likelihood a nervous one. In organic cases the symptoms will increase in intensity. Nervous cases are also distinguished by the mental depression, over-anxiety over trivial matters with a tendency to hypochondriasis and introspection.

Simple Hyperchlorhydria. As in nervous dyspepsia, a diagnosis of simple hyperchlorhydria or hyperacidity should never be made until all organic conditions, e.g. duodenal/

duodenal ulcer have been excluded. Although hyperchlorhydria is a grave symptom of an organic lesion, it is in many cases a secretory neurosis and should be labelled apart as a special clinical condition. In duodenal ulcer the symptoms are definite, occult blood is usually discernible in the faeces, and pain is in proportion to the quality and quantity of the food ingested; while in hyperchlorhydria the symptoms are more irregular in every way, occult blood is never found with it, and pain is not as a rule proportionate to the quality and quantity of the food taken. These features, plus the physical signs of ulcer of the duodenum, will help in discriminating this condition from tht of simple hyperchlorhydria.

Splenic Anaemia (Banti's Disease) is characterised by the blood picture of a persistent anaemia of the secondary type, with an enormously enlarged spleen and bleeding from mucous membranes; which features will serve to distinguish this disease from gastro-intestinal conditions such as duodenal ulcer. There is, in addition, hepatic enlargement in splenic anaemia.

Banti's disease should be kept in view in conditions where the typical digestive troubles are inconspicuous or absent, associated with severe haematemesis/

haematemesis or melaena.

Haemophilia is another blood disease which occasions error in diagnosis; as exemplified by a case of Moynihan's ("Duodenal Ulcer", p. 156)[46], on which he operated with the result that the patient died by reason of the uncontrollable haemorrhage from both the wound and the intestine. Haemophilia is characterised by its prominent hereditary history, combined with the fact that haemorrhages occur as the result of slight traumas, such as an abrasion.

Acute Thoracic Conditions. Abdominal affections, such as perforation in a duodenal ulcer, may be simulated by an acute attack of pleurisy or pneumonia, or even of bronchitis. It is particularly so in the case of a diaphragmatic pleurisy, in which the onset is dramatically sudden, the pain probably being referred almost exclusively to the abdomen, with supreme tenderness over the abdominal surface, rigidity of abdominal wall, etc. In illustration, I here relate my own experience in the rôle of patient. On the 28th July of last year, in Travancore (S. India), while driving my motor car, I was, without any previous warning suddenly seized with an acute lancinating pain, localised to a spot below the right nipple near the tips of the ninth and tenth ribs at the junction with their cartilages, the pain/

pain being aggravated on movement and in taking a full breath. I was practically disabled in the right arm and side, but managed to drive home (after covering 48 miles) by steering with my left arm and by keeping my right arm close to my side. I had to lie up on my arrival, and by the evening the temperature rose to 100°F; the pain was now agonising. I placed myself in the hands of my senior subordinate Dr. E. Poonen, who called in his colleague Dr. P. N. Lakshmanan. The latter remarks, under date 1st August, 1911, "I arrived at 7 a.m. and found the patient bathed in sweat and with a temperature of 99.2°F. He had a rigor at 5.30 that morning and the temperature had risen to 102°F. He had been given 10 grains of quinine by the mouth. He lay on his back unable to move; respiration costal, about 34 per minute, and any attempt at deep inspiration or a cough caused him agony on the right side. The abdomen was fixed, no movement there being discernible. Liver dulness normal. No pain or tenderness over the region of the gall bladder. On auscultation friction was distinctly heard during inspiration and partly during expiration over an area covered by a rupee one inch below the level of the right nipple and between the right anterior axillary and mammary lines. Palpation over this part was painful and there was very great tenderness/

tenderness over the tip of the tenth rib near its junction with its cartilage, any pressure on which was unbearable. Percussion revealed no appreciable dulness over the area of friction. Digestive system - tongue covered with a dirty white fur. There is pyorrhoea alveolaris in connection with the incisors of the upper jaw. No nausea or vomiting. Bowels constipated and moved only by an aperient or the enema. Circulating system - pulse 84 per minute regular and strong. Nothing morbid. Muscular, cutaneous, and nervous systems - nothing abnormal. Urine - specific gravity 1022. Reaction acid. Somewhat high coloured. No albumin, sugar, bile acids, or pigments. Some urates present. Patient occasionally, even in health, complained of a dragging pain in the right lumbar and iliac regions. There is some tenderness over McBurney's point.

2nd. August. The temperature rose to-day to 103°F. at 3 p.m. and he had a rigor at 1.30 p.m.. He is constantly bathed in sweat night and day. Pulse 90, respiration 34, abdomen tympanitic, no motion or flatus passed. The blood was examined by the hospital bacteriologist and myself, and we failed to discover any malarial parasite or pigment.

3rd August - No rigor to-day; temperature kept at 100.4°, respiration 30, pulse 84; sweating continues/

continues, bowels relieved by an enema, the flatus tube had to be passed twice as the distress from tympanitis was great. He had two injections of acid hydrobromate of quinine of 5 grains each to-day. The blood was examined for differential count and reported normal.

During the next four days other symptoms developed which entirely masked the original picture.

4th August. Two rigors to-day, one at 7 a.m. and the other at 8.30 p.m., and the temperature rose to 103.4°; pulse 90, respiration 30. The rigor each time was followed by great depression and weakness. Two injections of quinine were given with 5 m. of Liq. Strychninae. Bowels relieved by an enema; the flatus tube had to be passed thrice. There was irritability of the bladder with much strangury and frequency of micturition. Friction sound continues to be heard over the area noted above but resonance not appreciably impaired.

5th August. One rigor at 10.10 a.m. Temperature 101°. Sweating continues day and night. Abdomen distended, motion not passed, an enema of saline and asafoetida brought only water and some gas. The tympanitis intensified the suffering. Flatus tube resorted to thrice gave no relief. A glycerine enema was more successful. No nausea or vomiting. To-day/

To-day another physical sign was noted. There was a marked fulness, amounting to a prominence, and dulness on percussion over an area covered by the palm, extending from ½ inch below the line joining the right anterior superior iliac spine with the umbilicus, downwards and towards the median line.

6th August - A feeling of chill at 9.30 a.m., temperature rose to 102°F., pulse 81, respiration 26 (still shallow and catchy), deep inspiration impossible; abdomen continues to be fixed. My senior colleague and I discussed the matter and came to the conclusion that the temperature, the rigors, the rapid weakening, the perpetual sweating and tympanitic distension of the abdomen, all pointed to a focus of acute suppuration probably in the region of the appendix, and that it may be one of those cases where the appendix stretched across the brim into the pelvis where it may have become adherent to the bladder (thus accounting for the irritability) or it may have something to do with the liver or both, and the existence of pyorrhoea alveolaris lent colour to this view. It was thought advisable to call for another opinion, and Dr. Davidson (M.D. Edin.) of Neyoor Hospital was wired for.

7th August. Dr. Davidson arrived. He also came to exactly the same conclusion. Delay he thought was not/

not justifiable and the following day was fixed for the operation."

Of the further course of events, Dr. Davidson says in his notes - "Major Bidie was clearly in a bad condition. For a week his temperature had not touched the normal and was 101° at the time of examination. There was much prostration, and the patient was bathed in perspiration. There was marked tympanitic distension of the abdomen, and a tender dull area more or less in the appendical region. There was also a tender area over the lower part of the liver, where friction could be heard on auscultation. The breathing was shallow and catchy. Deep inspiration was difficult or impossible; and to turn even on the side caused a tendency to syncope. The history of vesical irritability, the rigors, the constant temperature, and the present condition of absolute prostration made us feel that something was clearly necessary to clear up the diagnosis, if at all possible. The possibility of hepatic abscess, empyema, or appendical or caecal abscess all presented themselves to our minds. Peritonitis seemed almost certain. After careful consideration of all the clinical facts, operation was agreed upon, with a view of examining the appendical region by laparotomy and the hepatic area by needle puncture. Operation - This was done on 8th August, 1911 - Dr Lakshmanan operating/

operating and I rendering what assistance was necessary. Dr. Poonen was also present. A large amount of urine was first drawn off, and the dull area in the appendix region cleared up. We decided, however, to go on. The appendix was examined, and did not appear to be markedly inflamed or adherent. The wound was closed, and then needle puncture of liver performed. No pus was found. While disappointed in not finding a more definite cause for all the various serious symptoms, we felt relieved to know that there was no pus in the caecal or hepatic regions. The temperature, however, fell at once after the operation and remained normal, though the patient rallied but slowly. When I saw the patient a week or so later, he was progressing well, but still in a very weak condition, hardly able to turn in bed without assistance. Diagnosis - Malaria, I think, can be put out of account. Tubercle, unfortunately, was difficult to absolutely eliminate, but all that could be done in the way of tests was tried, and nothing positive was obtained. My opinion is that probably the patient got a severe chill when motoring, and that he had inflammation in several regions at the same time. The pain on right side of chest was probably of a pleuritic nature (diaphragmatic very likely), but I still feel that there was (or had been prior to the operation) a good deal of local/

local peritonitis somewhere in the abdomen, which led to the tympanitis and the bladder retention." *

Similar cases are recorded by Barnard ("Lancet", 1902, ii, 280)[47], and by Richardson ("Boston Med. and Surg. Journal", 1902, i, 399)[48], illustrative of the practically perfect mimicry of acute abdominal conditions by disease above the diaphragm.

When dealing with such cases, particular attention should be given to the following points in framing an opinion :-

(i) The temperature is of all-importance, for it is a matter of clinical experience that rarely does the temperature rise to 102° (or over) in an acute abdominal lesion; whereas in acute thoracic affections, the temperature may fluctuate between 103° and 105°.

(ii) The pulse and respiration rates are disproportionate. In acute thoracic conditions, the pulse seldom ranges above 100, and the respirations between 35 and 45 per minute; whereas, in abdominal lesions, a pulse rate of 120 and a respiratory rate of about 25 are usually met with.

(iv) In chest affections, the resistance of the abdominal wall fluctuates, while its tenderness to pressure is superficial. In abdominal conditions, on the contrary, the abdominal wall remains consistently rigid/

*See appendix (5), p. 85.

rigid, and tenderness extends deeply.

(v) In cases of diaphragmatic pleurisy, the movements of respiration are exaggerated in the upper costal area, with comparatively quiet respiration in the lower part.

I will now bring this thesis to a close by briefly summarising the various points dealt with, with an expression of my opinion as to the conclusions arrived at.

Personal experience, supplemented by clinical data, amply proves the fact that the condition known as ulcer of the duodenum is of much more frequent occurrence than was until recently realised, while the following typical symptoms are at one time or another usually met with in cases of this disease:-

(1) A sensation of fulness (of being "blown out"), accompanied by epigastric distress, after taking food, with gaseous eructation and signs of gastric hyper-acidity (e.g. "water brash" in the mouth, etc.).

(2) Pain of a particular kind - termed "hunger pain" - which cannot be considered as pathognomonic of duodenal ulcer. This contention is fully borne out by clinical evidence.

(3) Localisation of a tender area, two or three inches/

inches in diameter, slightly above and to the right of the umbilicus.

(4) Presence of haematemesis, melaena, or "occult blood".

(5) Gastric dilatation. Due in the commencement to a so-called "protective spasm" of the pylorus, having the object of delaying the passage of the over-acid contents of the stomach into the ulcerated duodenum. Cicatrical contractions and adhesions later in the disease, produce dilatation of the stomach from actual organic stenosis of the pylorus. The recognition of dilatation in the early stage is very desirable.

In gastric ulcer (unless the pylorus be also implicated), the stomach is usually contracted in a small degree, while the sphincter relaxes at an early period of digestion with the object of allowing the passage of the gastric contents, thus affording temporary relief.

The importance of establishing an accurate diagnosis is manifest from the following facts:-

(1) It will primarily be conceded that ulcer of the duodenum is a condition of considerably greater gravity than ulcer of the stomach.

(2) Secondly, that the liability to perforation is much greater in duodenal ulcer (as compared with gastric ulcer); and that the probability of such a lesion/

lesion having taken place imperatively calls for immediate relief by surgical measures. Some cases recover without operation, as witness the following recorded by Alexander Miles ("Edinburgh Medical Journal", Aug. 1906, p.113)[49]:-

About two years ago, I saw, with Dr. James Mill, a patient aet. 31, who had for some time previously suffered from symptoms of duodenal ulcer. Two days before I saw him, while helping to lift a heavy box, he was noticed to become"deadly white", and he looked so ill that his fellow-workmen sent for brandy, and gave it to him. He had not been conscious of anything giving way at the moment, but a short time afterwards he was seized with intense pain in the pit of the stomach, doubling him up and causing him to cry out. On reaching his home, he was in a state of profound shock, with a markedly sub-normal temperature, and a pulse of 45. There was pain all over the abdomen, and an area of great tenderness to the right of the epigastrium. He passed a very bad night, but next morning began to improve, and as the improvement was maintained he was not operated upon. The opinion we formed was that the muscular strain had determined rupture of a duodenal ulcer, which had set up a localised peritonitis. In the course of a few weeks he recovered completely.

(3)/

(3) Thirdly, that the haemorrhage, which occurs in cases of duodenal ulcer, is in all likelihood of greater danger, from its profuseness, than in gastric ulcer. It is worthy of note that haemorrhage associated with perforation is rare.

(4) Can a permanent cure be effected in ulcer of the duodenum by medical means? Certainly; but the percentage of permanent cures is less than in cases of ulcer of the stomach. Healed duodenal ulcers have been found at the autopsy, and undoubtedly a certain proportion of cases, (which have exhibited classical symptoms), have completely recovered without surgical interference. However, the danger lies in the subtlety of the condition, in that all symptoms may vanish only to recur later. This feature of a tendency to recurrence I have already dwelt on as very characteristic of the disease. How can we account for this peculiarity of recurrence? I think the explanation is found in the fact that a multiple ulceration or the appearance of a fresh ulcer takes place independent of the primary lesion. For, has it not been noted, in cases suggestive of an ulcerative dyscrasia, that, in addition to the duodenal lesion, ulceration of the mouth and oesophagus have been present. We may therefore conclude that operative interference, far from combating this dyscrasia or affording/

affording relief to the true cause of the lesion, may even supply a damaged mucosa inviting renewed ulceration in an individual thus pre-disposed.

(5) In what class of cases should we have recourse to surgical interference? Those in which, after careful medical treatment for several weeks, no permanent benefit results; and when, after conscientious study of all the features, one feels that there is an organic lesion present. Under such circumstances, one should have no hesitation in advising a laparotomy. The following points are helpful in arriving at a decision:-

1. The possibility or likelihood, in any given case, of the failure of medical treatment.

2. A possibility, or it may be, an even chance that the patient may have a relapse if temporarily cured.

3. The chance of development of pyloric stenosis.

4. A risk of perforation or of severe haemorrhage occurring.

5. The danger of the development of cancer in the site of the ulcer.

6. Various conditions resulting from adhesions, such as a crippling of the action of the stomach, obstruction of bile and pancreatic ducts, kinkings (productive of much pain and other disturbances) etc., often/

often calling for late operation.

7. Dangers from a troop of late complications of slowly developing perforation; among which one may encounter peritonitis, subphrenic abscess, pyo-pneumothorax, empyema, pneumonia, perforation through the lung, septic pericarditis, mediastinitis, pancreatitis, suppurative processes about the liver and gall bladder, and general septicaemia. Cases are unfortunately reported under such headings without a recognition of the origin of the process.

8. The development of a marked degree of anaemia and malnutrition, from disturbance and crippling of the digestive apparatus, as to lead to neurasthenia, hysteria, and other functional nervous conditions, or even pave the way for a ready and it may be fatal infection of microbic origin.

The results aimed at by an operation are :-

(1) Duodenal rest.

(2) Prevention of mechanical irritation by food.

(3) Provision of a second outlet for the passage of the food.

(4) Neutralisation of gastric contents, or their outlet before maximum acidity is reached. The usual experience is the speedy relief of all signs of acidity.

(5)/

(5) Relief of pain; practically a certainty in the early post-operative stage.

(6) Obviation of the risk of haemorrhage and perforation.

(7) Chances of permanent cure and prevention of recurrence.

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1.

APPENDIX.

Moynihan ("Duodenal Ulcer", 1912, p. 142)[52] records a case of gastric ulcer mistaken by him for an ulcer of the duodenum. He says "In a case operated upon in 1910 I had made a diagnosis of duodenal ulcer after eliciting what I felt sure was an authentic history of duodenal ulcer. At the operation no ulcer was discoverable in the duodenum. In the stomach, on the posterior wall, nearer the cardia than the pylorus, was an ulcer, almost circular and two inches in diameter, deeply eroding the pancreas. The base of the ulcer was formed by the pancreas, the stomach wall having been thoroughly destroyed. Even after the recovery of the patient from the operation (excision of the ulcer, &c) the only discrepancies between the story given by him and the typical history, were that the seasonal variations had never been observed, that pain in the back had been unremitting, and that the symptoms had been present without intermission for over three years."

An excellent example of mimicry of the symptoms of duodenal ulcer by a diseased appendix is given by Mitchell ("Trans. Ulster Med. Soc.," 1910-1911)[53]:- "A young married lady, aged 29, visited me on the 28th of February, 1910, accompanied by her husband. She informed me she had come to arrange for an operation for duodenal ulcer. She had seen several medical men, had tried milk diet and rest in bed, without benefit. Her daily history with slight variations was as follows: Quite comfortable when she got up in the morning; breakfast 9 o'clock, pain coming on about 11.30 to 12 o'clock, and getting worse till dinner at 1 o'clock. This meal at once gave relief, and she was quite comfortable till between 3 o'clock P.M. and 5 o'clock P.M., the interval depending on the nature of her dinner. Once the pain began it steadily got worse, so that if she happened to be down town shopping she was obliged to have afternoon tea, the result of which was prompt relief. She took her ordinary tea about 6.30, after which she generally remained well, but occasionally the pain came back about 8 o'clock. It, however, never came on after she went to bed or wakened her at night. She had slight tenderness over the right rectus at the level of the umbilicus. This being below the usual site, I enquired whether she ever had an/

2.(contd.)

an attack of appendicitis, but she could recollect nothing of the kind. An operation, 6th of March, 1910, her family medical attendant being present, we found a healthy stomach and duodenum. The appendix was $4\frac{1}{2}$ inches long. At a point 1 inch from the caecum it was tightly constricted, the remaining $3\frac{1}{2}$ inches was distended to the thickness of an index finger, and the tip was fixed by adhesions. The appendix was removed. She made rapid recovery, and her medical attendant informs me she has remained well since the operation.

The history at first sight appeared quite typical. The absence of night pain and the position of such tenderness as appeared to be present suggested the posibility of an appendical origin. Our incision was therefore made through the right rectus opposite the umbilicus, so that it could be extended up or down as required."

3.

Moynihan ("Brit. Med. Jour.," 1911, i, 733)[54] records an instance of acute pancreatitis, which he mistook for a perforated ulcer of the duodenum; the patient being a fat man, inclined to the free use of alcohol, with a lividity of countenance and to a less amount of the body. The diagnosis was cleared before the incision of the peritoneum, by the discovery of fat necrosis in the subperitoneal fat.

4.

The following case of carcinoma, developing in the base of a chronic ulcer, is recorded by Peck ("Annals of Surgery", 1910, i, 952)[55]:-

About two years ago Dr. Peck operated on an acute perforated duodenal ulcer which had come on after a sudden strain in lifting. There was no induration about the ulcer, and the patient made a good recovery. About a year later there was a second acute perforation, apparently at the same site, but this time the inflammatory changes surrounding the lesion were well marked. He simply closed the perforation, without doing a gastro-enterostomy. The patient recovered from the operation, but did not do well. There were symptoms of obstruction, and two weeks later he was forced to perform a gastro-enterostomy, from the effects of which the patient died. The autopsy showed a more extensive and deeper duodenal ulcer, with a very small carcinoma which must have developed in the interval since the first operation.

5.

The following case is recorded by R. T. Morris ("Trans. of Society of Alumni of Bellevue Hosp.," 1898-99; Dec. 7., 1898, p.55)[56] :- " A woman, 45 years of age, was attacked by symptoms of acute peritonitis. Temperature, 105°F; rigid abdominal muscles. Intense pain and local tenderness in right iliac region. She gave a history of previous lesser attacks of pain in the right inguinal region. On operation it was discovered that the appendix and right oviduct were bound together by adhesions. The appendix on examination was found to be normal. It had been caught in oviduct adhesions some time previously. On the following day the patient was found to have typical lobar pneumonia. She recovered. I was called to this case by one of the most expert diagnosticians in the city. A few days later he showed me a precisely similar case of lobar pneumonia with abdominal symptoms, in a young man for whom a correct diagnosis had been made."

6.

Mr. Caird ("Edin. Med. Jour.," April, 1911, p. 319) relates an illustrative case of the mimicry of some of the symptoms of duodenal ulcer by a malignant growth in the stomach:-

"Mrs. X., aet. 45, developed gastric symptoms in summer 1909. Epigastric pain, of a dull, gnawing character, came on 2½ to 3 hours after meals, and was invariably relieved by taking more food. This pain has persisted for the last eighteen months with varying severity, and latterly she has been able to control it by taking sod.-bicarbonate and Gregory's powder. She has been greatly troubled by nocturnal pain, and has been in the habit of taking some form of nourishment - e.g. soup - at 2 A.M., and she frequently eats biscuits in the morning about one hour before breakfast to stave off the epigastric pain. There has been no vomiting. She is fairly well nourished, but has lost much weight.

From a general consideration of this case, and that no epigastric resistance was palpable, and that the test meal contained no free HCl, the correct diagnosis of carcinoma was made. The disease involved the lesser curvature and pyloric antrum, and was at least one inch remote from the pylorus."